

EVALUATION AND MANAGEMENT OF THE DIZZY PATIENT

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Balance in man is a sophisticated and complex mechanism comprised of sensory inputs from the vestibular apparatus, vision, and proprioception. These pass into the central nervous system (CNS), are integrated and modulated by activity of the cerebellum, the extrapyramidal system, the limbic system, and the cerebral cortex, and provide perception of head and body position in space, eye movement control, and appropriate static and dynamic postural function. Alterations in the sensory inputs, integrating mechanisms, or effector organs can result in the perception of dizziness or vertigo, disordered eye movements and disequilibrium, or instability. A wide variety of pathological processes may give rise to dizziness, such that patients present to many different specialists, but most commonly to the ear, nose, and throat (ENT) or neurology departments (table 1).

Dizziness is extremely common, both in primary care and at the tertiary level, and by the age of 60 years, one third of the population has suffered from a balance disorder. While in primary care many cases of dizziness resolve spontaneously, in tertiary care dizziness is commonly associated with significant morbidity and, in the older population, if compounded by falls, mortality. Thus, a clear diagnostic strategy including a detailed neuro-otological examination, as outlined in the previous article, is essential if an accurate diagnosis is to be made. Diagnosis is key to the rehabilitation and management of the dizzy patient.

Three main groups of disorders giving rise to disequilibrium can be identified—general medical, neurological, and otological—with a few other disorders such as visual vertigo, cervical vertigo, and the multisensory dizziness syndrome in the elderly, falling outside this classification. A detailed history and examination, as outlined in the preceding section, will usually point the examiner in the correct direction for appropriate investigation. Inevitably there is some overlap (fig 1), in as much as diffuse cerebrovascular disease may produce both neurological and neuro-otological abnormalities, while general medical disorders, such as diabetes mellitus and autoimmune syndromes, may give rise to both labyrinthine and/or central vestibular dysfunction.

Patients with persistent dizziness/vertigo/disequilibrium and evidence of vestibular dysfunction on standard vestibular tests (see Davies, p iv32) fall into two main categories: those with specific diagnoses (for example Menière's disease and benign positional paroxysmal vertigo), for whom there are standard established treatment regimens, and those with peripheral labyrinthine pathology, in whom spontaneous adequate vestibular compensation does not take place, and chronic vestibular symptoms become the overriding clinical presentation requiring management.

The scope of this article does not allow detailed consideration of visual, neurological or general medical causes of dizziness, and will concentrate on the management of patients with peripheral vestibular disorders and the general strategy for managing patients with neurological disorders manifesting with vertigo.

PERIPHERAL VESTIBULAR DISORDERS AND COMPENSATION

Epidemiological studies providing data on causation in “the dizzy patient” all highlight the overriding importance of peripheral vestibular pathology, whether the patients present to an otolaryngologist or a neurologist. The balance system in humans has a remarkable capacity to adapt and to learn new behaviours, and this is characterised by the rapid symptomatic recovery following peripheral vestibular pathology, referred to as “vestibular compensation”. This is the collective term given to a variety of processes including adaptation/habituation, leading to recalibration of the gain of the vestibular reflexes and substitution of both sensory inputs and motor responses, together with alteration of strategies used for balance (fig 2). This process is independent of the causation of vestibular dysfunction, and the components of vestibular compensation vary in their efficacy in facilitating symptomatic recovery of perception, oculomotor and postural stability from subject to subject. Thus, some patients recover stability with no head movement induced vertigo, but may suffer from pronounced visually induced dizziness, while others experience pronounced instability, but little disordered perception of imbalance (that is, dizziness).

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Table 1 Causes of dizziness

General medical	Otological
<ul style="list-style-type: none"> Haematological Anaemia Hyperviscosity Miscellaneous Cardiovascular Postural hypotension Carotid sinus syndrome Dysrhythmia Mechanical dysfunction Metabolic Hypoglycaemia Hyperventilation 	<ul style="list-style-type: none"> Menière's disease Post-traumatic syndrome Positional nystagmus Vestibular neuronitis Infection Otosclerosis and Paget's disease Vascular accidents Tumours Auto-immune disorders Drug intoxication
Neurological	Miscellaneous
<ul style="list-style-type: none"> Supratentorial Epilepsy Syncope Psychogenic Infratentorial Multiple sclerosis Ischaemia Infective disorders Degenerative disorders Tumours Foramen magnum abnormalities 	<ul style="list-style-type: none"> Ocular Cervical Multisensory dizziness syndrome

spontaneous nystagmus with the fast phases directed away from the affected ear, and postural instability (ataxia). These symptoms are related to the differences in the levels of tonic activity in the central vestibular nuclei, resulting from the vestibular pathology. *Static* oculomotor recovery is a robust process that starts 3–4 hours after the onset of the lesion, and is complete in a few days. It occurs in parallel with the reappearance of resting neural activity in the ipsilateral vestibular nuclei and “rebalancing” of the vestibular nuclei. Postural recovery, on the other hand, appears to rely more on propriospinal mechanisms. *Dynamic* symptoms are consequent upon abnormalities of the gain, symmetry, and phase of the vestibular reflexes, and recovery continues over months or years, being faster for postural than for oculomotor symptoms.

FAILURE OF COMPENSATION AND DECOMPENSATION

The majority of patients will “compensate” and function virtually normally within six weeks to six months following a peripheral vestibular disorder, although this symptomatic recovery does not parallel recovery of vestibular function. Some patients demonstrate incomplete resolution of symptoms, with persistent failure of recovery from the initial event, while other patients recovery normally, but then demonstrate recurrent episodes of decompensation, with episodes of vertigo interspersed with periods during which they are asymptomatic. The reasons for incomplete

Damage to the vestibular elements of the peripheral labyrinth results in a characteristic syndrome of acute vertigo,

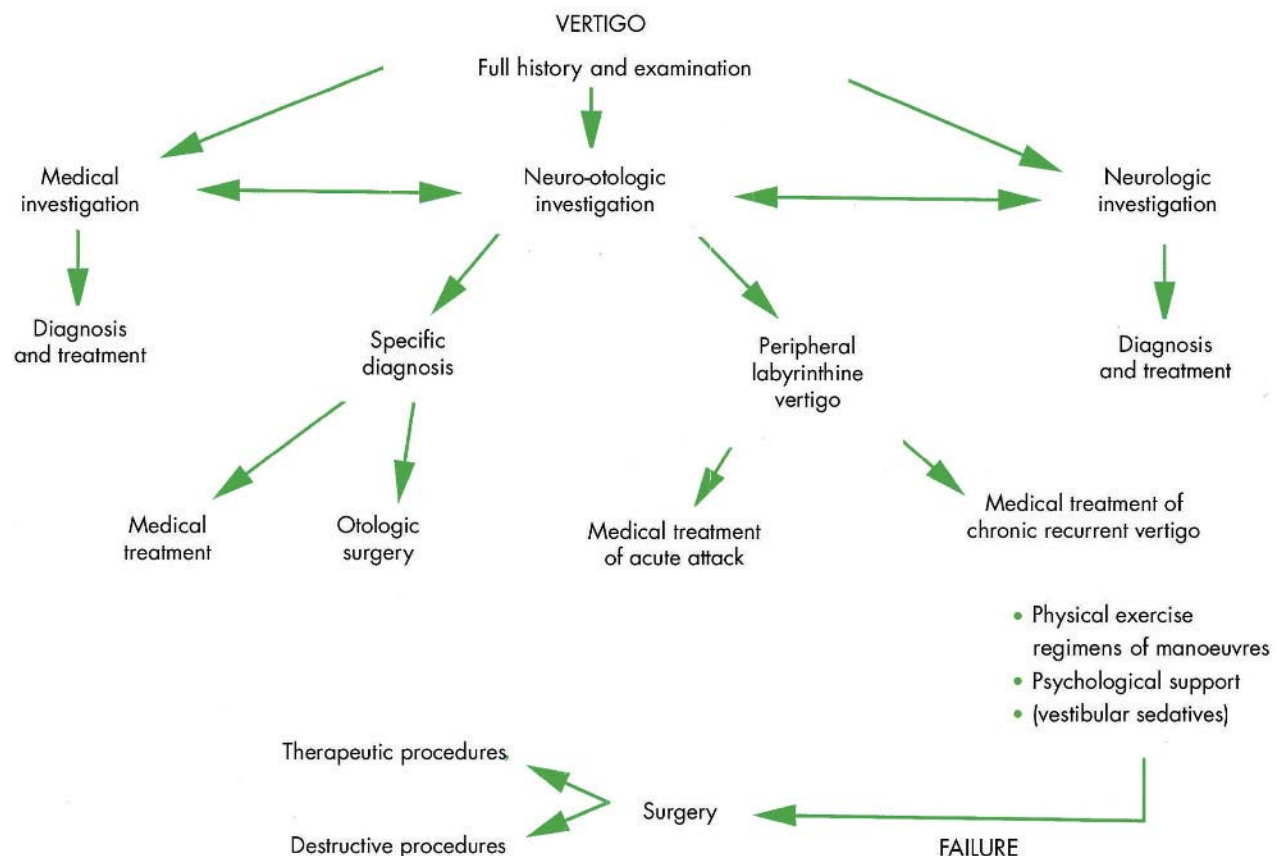


Figure 1 Outline strategy for management of vertigo. Reproduced with permission from Luxon LM, Davies RA, eds. *Handbook of vestibular medicine*. London: Whurr Publishers, 1997.

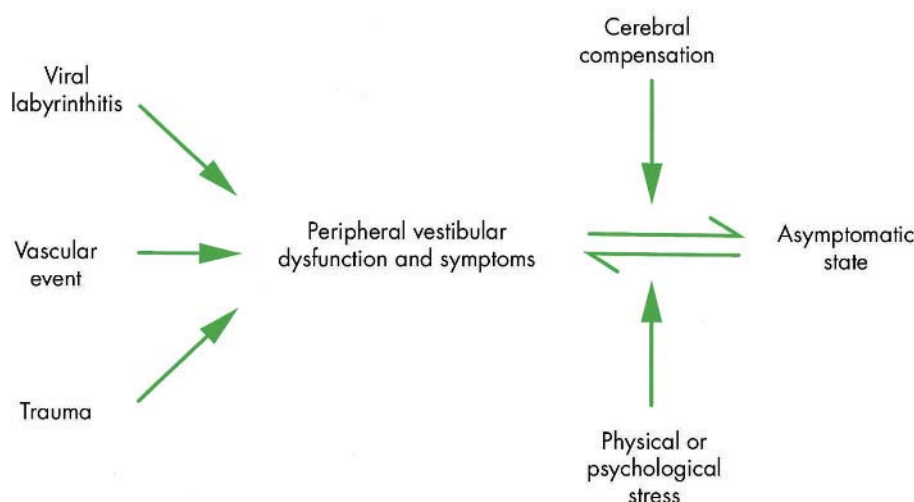


Figure 2 Diagram of the natural history of peripheral vestibular pathology. Reproduced with permission from Luxon LM, Davies RA, eds. *Handbook of vestibular medicine*. London: Whurr Publishers, 1997.

asymptomatic recovery may be single or multiple (fig 3), and evaluation of these factors is crucial in the management of a patient with a peripheral vestibular disorder (see Davies, p iv32) and persistent chronic symptomatology. The most common causes of failure of compensation are psychological disorders, impairment of the other sensory inputs required for balance (vision/proprioception), use of drugs with action upon the central nervous system (importantly including vestibular suppressant drugs), and co-morbid systemic disorders.

TREATMENT OF THE VESTIBULAR PATIENT

Successful management of the patient with dizziness depends upon accurate diagnosis, an understanding of vestibular physiology, appropriate intervention strategies, and the physician's awareness of the overlap between vestibular, autonomic, and psychological aspects of vestibular pathology.

Appropriate management for the dizzy patient of vestibular pathology results in the ability to control and coordinate eye, head, and body movements in order to maintain gaze,

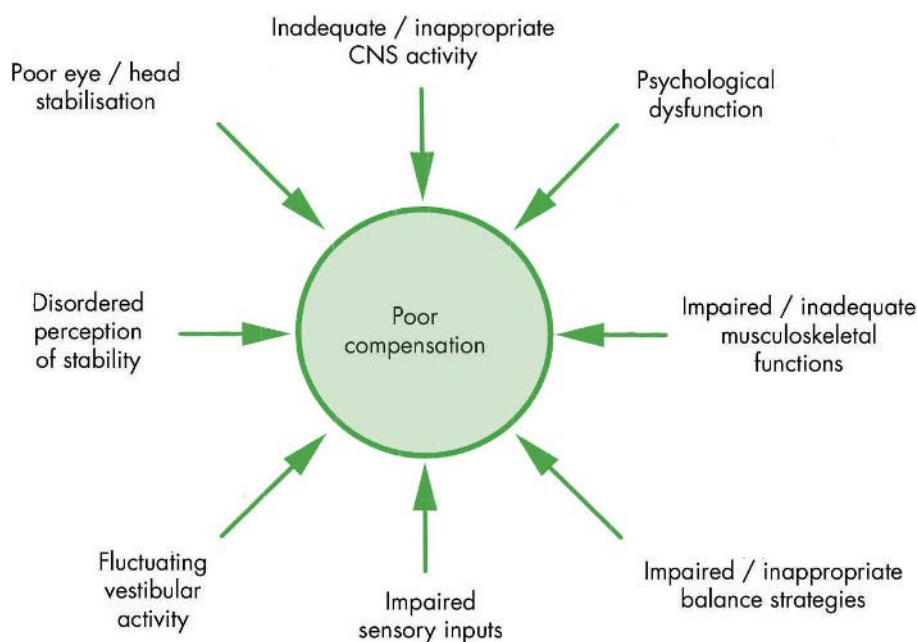


Figure 3 Factors affecting compensation. Reproduced with permission from Luxon LM, Davies RA, eds. *Handbook of vestibular medicine*. London: Whurr Publishers, 1997.

stability, and posture. Management also aims to achieve appropriate vestibular perception without adverse symptoms, despite the persisting dynamic asymmetry in the gain of vestibular reflexes. Evaluation and efficacy of intervention are often difficult due to the dysynchrony between vestibular symptoms and signs and vestibular test results, and treatment should include validated psychological questionnaires (in view of the high prevalence of psychological disorders in patients with chronic vestibular symptoms), together with questionnaires evaluating vestibular symptomatology, disability, and handicap. Objectively, posturography results may provide direction for intervention in terms of balancing strategies, and additionally provides objective quantification of increasing stability.

There are five main arms of management intervention:

- ▶ general medical evaluation, with correction/amelioration of associated morbid conditions
- ▶ pharmacological intervention
- ▶ vestibular rehabilitation with physiotherapy and specific physical manoeuvres for the management of benign positional paroxysmal vertigo
- ▶ psychological intervention
- ▶ surgery.

On the basis of the diagnosis, an appropriate rehabilitation plan should be constructed for each patient, and should be explained in detail to ensure understanding and active compliance with the programme (table 2).

General measures

In the dizzy patient, a general medical examination should identify co-morbid, systemic conditions such as hypertension, vascular disease, diabetes, autoimmune disorders, and psychological pathology, all of which may impact upon vestibular compensation if appropriate treatment is not in hand. Specifically, ophthalmological and rheumatological/orthopaedic problems should be addressed, to ensure optimal visual and proprioceptive input for vestibular rehabilitation.

Pharmacological treatment

Acute vestibular symptoms caused by either peripheral or central vestibular disorders may be helped by use of anti-emetic and vestibular suppressant drugs. Despite our current understanding of vestibular neurochemistry, treatment of vestibular disorders remains mostly empirical, often with relatively poor understanding of the specific antivertiginous action of any particular drug, and a lack of appropriate clinical trials establishing efficacy, duration, and dosage of the drug. The ideal antivertiginous drug would suppress

dizziness, prevent vomiting, and promote vestibular compensation, but currently no such drug is available.

The drug treatment of a vestibular disorder is initiated for one of three main reasons:

- ▶ treatment of the acute vestibular symptomatology
- ▶ specific treatment of a condition that causes vestibular symptoms—for example, Menière's disease, migraine or epilepsy
- ▶ non-specific but empirical treatment of a chronic vestibular disorder—for example, central vestibular symptomatology.

Symptomatic treatment of acute vestibular episode

Symptoms of vertigo, nausea, vomiting, sweating, pallor, and diarrhoea are extremely alarming and debilitating to a patient, who commonly suspects that the symptoms are life threatening. The initial step in the management of such a patient must be to reassure and explain the nature of the symptoms, together with providing hydration if necessary. Antiemetics should be administered—for example, hyoscine, prochlorperazine, promethazine, cyclizine, dimenhydrinate, and metoclopramide—orally if feasible, intramuscularly, as a suppository, or via the buccal membrane. Hyoscine administered transdermally and prochlorperazine administered via the buccal membrane have been shown to be potent antiemetics, with some evidence of efficacy in the suppression of vertigo and/or dizziness.

Calcium channel antagonists, cinnarizine and cyclizine, have vestibulosuppressant effects, although cinnarizine is less effective than hyoscine in controlling patients' sickness. Both drugs may give extrapyramidal side effects, and should be used only briefly in older patients.

Diazepam has no specific action on the vestibular system, and acts by reducing neural activity and causing inhibition throughout the CNS, including activity in the vestibular nerve and vestibular nuclei. The role of this drug in the treatment of vestibular disorders is controversial, but it is widely used for its anxiolytic activity in acute vestibular crises.

Specific treatment of vestibular disorders

Migraine affects approximately a fifth of the population, is the most common cause of dizziness/vertigo in children, and is a common presentation both with headache and in the headache-free periods in adults with migraine. Vestibular test results are commonly normal, although approximately 20% of cases demonstrate a canal paresis on caloric testing. Diagnosis depends on the history based on the International Headache Society criteria. The treatment of migraine associated dizziness parallels the treatment of migrainous headache. General measures include dietary restriction, lifestyle adaptations, stress reduction techniques, and vestibular rehabilitation in the presence of a fixed vestibular deficit. The course of treatment includes simple over-the-counter analgesics, triptans, ergot derivatives, and acetazolamide. Prophylactic medication includes β blockers, calcium channel blockers, serotonin reuptake inhibitors, and amitriptyline.

Episodic ataxia, while rare in comparison with migraine, may present with acute vertigo and ataxia, with or without interval symptoms, in both adults and children. Both acetazolamide and 4-aminopyridine are effective in episodic ataxia type II.

Menière's disease is perhaps the most commonly misdiagnosed vestibular condition. Diagnosis should be based on

Table 2 Rehabilitation programme

1. Investigation and diagnosis
2. Explanation
3. Rehabilitation plan
 - correction of remediable problems
 - general fitness programme
 - physical exercise regimens
 - psychological assessment
 - medication
 - realistic family/social/occupational goals
4. Monitoring/feedback/follow up
5. Discharge

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strict criteria as defined by the American Academy of Ophthalmology and Otolaryngology (AAOO) committee on hearing and equilibrium guidelines. The treatment of Menière's disease remains controversial and empirical, not least because of the continuing quest to define the exact underlying pathogenesis of this condition. There are few double blind randomised studies assessing treatment efficacy, and it is important to recall an 80% placebo response in this condition. Medical treatment aims to influence underlying pathology of endolymphatic hydrops or the postulated immunological pathogenesis of this disorder, while destructive surgery may either be medical (intratympanic injection of aminoglycosides) or surgical (see below).

Clinical treatment normally includes institution of a low salt diet, although there are no double blind trials reporting efficacy of this regimen. Notwithstanding this, the author's experience, like that of many other clinicians, is that strict adherence to a low salt diet, together with diuretics, is a highly effective strategy in patients who can be persuaded to follow this regimen. Patient compliance depends upon education about unexpected levels of salt in many foods (for example, cornflakes, prepared foods, and tinned food). Diuretics have been reported in a small number of double blind trials to be effective in the long term control of vertigo, but not of auditory symptoms. The most common prescription is for bendroflumazide 10 mg each day, together with potassium supplements. Again, there are no data on the efficacy of this empirical treatment in Menière's disease. More potent loop diuretics such as furosemide (frusemide) should be avoided because of potential ototoxicity.

Betahistine is a histamine analogue which is proposed to have value in Menière's disease, as a result of improving micro-vascular circulation in the stria vascularis of the cochlea, thus reducing endolymphatic pressure. A number of trials of this drug are reported, but none adhere to the AAOO criteria. Thus, there remains no definitive evidence for its use.

Steroid treatment has been proposed both systemically and transtympanically in Menière's disease, on the assumption of an autoimmune pathogenesis. There are no double blind controlled studies demonstrating the clinical efficacy of steroids or other immunosuppressants, and their use remains open to question.

Management of Menière's disease by destructive surgical or medical treatments must be considered extremely carefully in view of the possibility of bilateral Menière's disease, with subsequent bilateral loss of auditory and vestibular function. Streptomycin and gentamicin have been used because of their selective vestibulotoxic effect, with gentamicin the drug of choice. There is no consensus on optimal protocol, technique of administration, or end point of treatment. Moreover, while this technique has been advocated in patients with intractable vertigo unresponsive to medical treatment with good auditory function, there are reports of deafness developing in up to 30% of treated cases.

Surgical intervention includes the theoretical prophylactic approach of endolymphatic sac decompression, but there is no firm evidence on the efficacy of this intervention. Destructive procedures fall into two categories: those that aim to preserve auditory function, such as vestibular neurectomy; and those in which the disease process has already caused profound hearing loss in addition to intractable vertigo. In the latter group, labyrinthectomy is advocated, if medical treatment has failed.

Chronic vertigo

It cannot be overemphasised that chronic vertigo on the basis of a peripheral vestibular disorder should not be treated with antiemetics and/or vestibular sedatives, as these drugs are reported to impair vestibular compensation, and thus delay recovery. Regrettably, in practice these drugs are commonly prescribed for chronic vertigo both in primary and tertiary care.

Central vestibular disorders occurring with demyelination, degeneration or vascular events are commonly associated with persistent disequilibrium, nausea, and eye movement disorders. Such disorders are usually associated with unpleasant perceptions of motion and vegetative symptoms for which drug intervention (clonazepam, carbamazepine, flunarizine) may be helpful.

VESTIBULAR REHABILITATION

The cornerstone of vestibular rehabilitation relies on the plasticity of the central nervous system, which enables reorganisation of the mechanisms subserving balance and "symptomatic vestibular compensation". When supervising servicemen's rehabilitation during the second world war, Sir Terence Cawthorne, an ENT surgeon, and Dr Harold Cooksey, a rheumatologist, observed that soldiers with balance disorders after head injury improved more rapidly if they were active and mobile, than if they were inactive and bedridden. They therefore empirically devised the vestibular exercise regimen known as the Cawthorne-Cooksey exercises. These formed the basis of the multiplicity of physiotherapy vestibular rehabilitation regimes currently available. In the 1970s and 1980s, scientists working on animal models of vestibular compensation demonstrated that visual input and motor activity are indeed crucial for symptomatic recovery from unilateral peripheral vestibular disorders. There is now also some evidence that vestibular exercises may be of value in more central lesions involving the vestibular nuclei and cerebellum.

As noted above, vestibular compensation relies on recalibration of the vestibular reflexes, and sensory and motor substitutions in terms of input and predictive activity. For this model to be effective, the vestibular disorder must be stable. In relapsing conditions such as Menière's disease, where there is fluctuating vestibular function, rehabilitation physiotherapy is unhelpful until intervention or spontaneous recovery brings about a stable vestibular situation. Thus, a correct diagnosis is essential to ensure appropriate treatment of conditions characterised by varying vestibular function—for example, migraine, benign positional vertigo of paroxysmal type, vestibular Schwannoma, or vascular disease.

Moreover, as noted above, the sensory inputs required for balance must be optimal, and thus if there are any remedial visual problems (for example, cataracts), or conditions which may affect proprioception (for example, arthritis/autoimmune disease), these must be optimally managed medically, in conjunction with vestibular rehabilitation programmes. Most importantly (see below), psychological factors play an important part in the effectiveness of vestibular rehabilitation therapies, and psychological disorders must thus be considered and treated where appropriate.

In each individual patient, it is helpful to define the strategy used for balance (visual, vestibular, or proprioceptive dominance) and the particular difficulties encountered by the individual. The rationale of the Cawthorne-Cooksey exercises is to stimulate each of the sensory inputs in a progressively

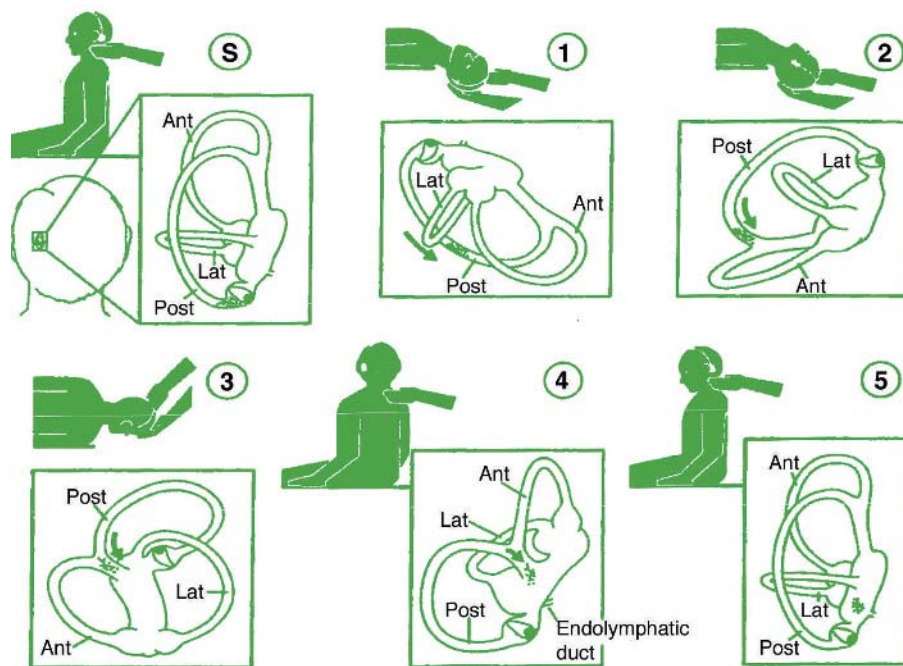


Figure 4 The Epley manoeuvre. Ant, anterior; Lat, lateral; Post, posterior. Reproduced with permission from Luxon LM, Davies RA, eds. *Handbook of vestibular medicine*. London: Whurr Publishers, 1997.

more difficult manner, to allow adaptation to occur. However, some patients have very specific symptoms, which are more amenable to “customised” exercises—that is, programmes are specifically structured on an individual basis to address the specific limitations and symptoms experienced by that particular patient. As compliance and active collaboration with vestibular rehabilitation programmes are required of the patient, such an individual approach may be more effective than a generic regimen. A detailed explanation of the mechanisms of balance and vestibular compensation is required for the patient to understand why physiotherapy should help them feel better from their dizziness/disorientation, rather than medication or an ear operation. There is some evidence in the literature that early intervention following vestibular pathology is associated with a better outcome. More surprisingly, recent work has shown that older people may compensate as effectively as younger people, and age per se is not a negative prognostic factor. Importantly, a general exercise regimen relevant to the age and ability of the patient should form part of the rehabilitation strategy.

PARTICLE REPOSITIONING PROCEDURES

In addition to systematic or customised exercise programmes for a peripheral vestibular disorder, a number of specific treatments aimed at treating benign positional vertigo of paroxysmal type (BPPV) have been developed. This condition is characterised by acute vertiginous episodes that are triggered by changes in the position of the head relative to gravity. Until recently BPPV had been attributed to cupulolithiasis—that is, degenerative debris adhering to the cupula of the posterior semicircular canal—making it gravity sensitive. However more recently, the hypothesis of canalolithiasis—that is, degenerative debris floating freely in the endolymph of the posterior semicircular canal—has gained

favour, as it explains more effectively the majority of the clinical features of BPPV.

Most commonly (93%), this condition affects the posterior semicircular canal, but rarely (5%) the horizontal canal may be involved, and extremely rarely (2%) the anterior canal. It is important that this condition is differentiated from central positional nystagmus (see Davies, p iv32), which is associated with neurological pathology. The initial management of this condition was habituation therapy with exercises, but in 1980, Brandt and Daroff proposed specific repetitive positional exercises based on the hypothesis of cupulolithiasis. Their initial report claimed a 98% success rate. However, more recently, specific repositioning manoeuvres (the Epley manoeuvre, the Semont manoeuvre) based on the hypothesis of canalolithiasis as the causation of BPPV have been introduced. These repositioning manoeuvres involve a series of specific consecutive head movements, which allow the debris in the canal being treated to gravitate out of the canal into the utricle, thus avoiding stimulation of the crista during head movements (fig 4). Most reports cite success rates of resolution of positional symptoms of between 80–95% following the first manoeuvre, although patients may experience vague disorientation for 2–3 days after treatment. Repeated manoeuvres, with mastoid vibration, may be necessary in a small percentage of patients, but currently it appears that less than 5% of patients cannot be improved by one of these manoeuvres. Provided the diagnosis is certain, it may be appropriate to consider surgical interventions such as plugging the posterior canal to bring about resolution of the positional symptoms in the small percentage of patients in whom medical management fails.

PSYCHOLOGICAL TREATMENT

The interaction of psychological factors in both the exacerbation of symptoms of peripheral vestibular disorder and

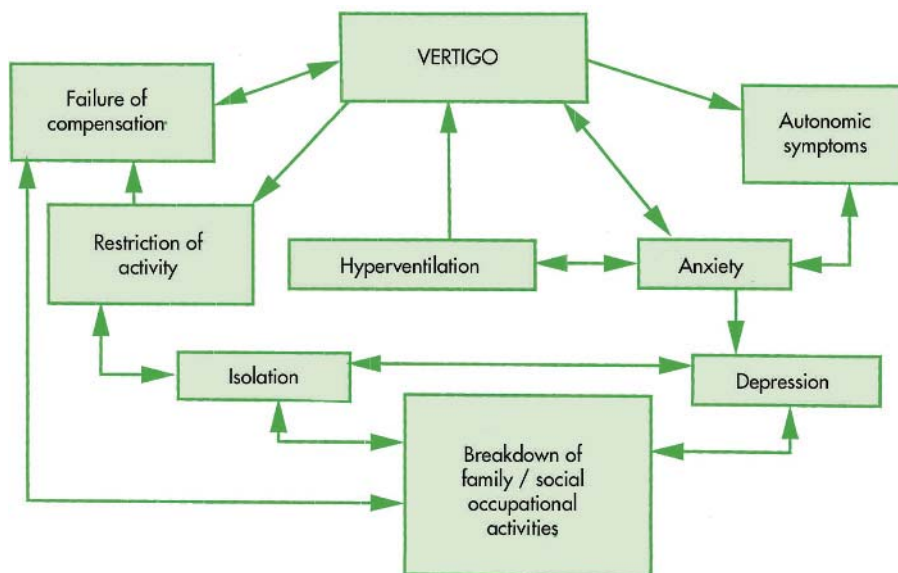


Figure 5 Diagram illustrating the interaction of autonomic, psychological, and vestibular symptoms.

failure of vestibular compensation cannot be overemphasised (fig 5). Many studies have highlighted the association of agoraphobia, avoidance behaviour, anxiety states, panic attacks, and depression with vestibular pathology. It is well recognised that in panic attacks autonomic symptoms and dizziness are common, but equally in vestibular disorders, anxiety and autonomic symptoms are common. Thus there is an intimate relation between these disorders and symptom complexes.

On initial assessment, it is helpful to consider psychological factors and specifically to ask about anxiety, panic attacks, and avoidance behaviour, together with mood change in a patient with dizziness. The presence of avoidance behaviour makes compliance with a vestibular physiotherapy rehabilitation programme unlikely and it is of value to address both cognitive and physical symptoms in parallel. More severe psychological disturbance should prompt immediate psychiatric referral, as vestibular recovery will not be possible until the psychological factors have been appropriately managed. In a young and otherwise healthy patient, who fails to compensate from an apparently straightforward peripheral vestibular disorder, the index of suspicion of an underlying psychological problem should be high.

SURGERY

It is now widely recognised that surgical intervention for vertigo is extremely rarely required. Specific exceptions to this rule include:

- ▶ life threatening complications of chronic middle ear disease
- ▶ neoplasia involving otological structures (for example, vestibular Schwannoma)
- ▶ trauma to the middle/inner ear (for example, a perilymph fistula).

In the past, therapeutic surgical intervention, particularly for Menière's disease, has been advocated, but there is little evidence that any of these procedures are more effective than medical management. Destructive surgical procedures must be undertaken with great care and very positive indications as outlined above. In a patient with a clear cut peripheral vestibular disorder, who is continuing to experience disabling

vertigo due to failure of compensation, there is no evidence to suggest that labyrinthine destruction will result in improved compensation.

"Failure" of medical management in Menière's disease often reflects inadequate attempts at medical management, or failure of patient compliance. It must be remembered that between 10–50% of cases of Menière's disease become bilateral, and, thus, labyrinthine destruction of one ear should, if possible, be avoided. Both vestibular neurectomy and transtympanic gentamicin—measures aimed at destroying vestibular function in Menière's disease, while preserving auditory function—may be effective, but both procedures carry a small but significant risk of profound sensorineural hearing loss. Surgical intervention may rarely be required for intractable benign positional vertigo not amenable to particle repositioning procedures, but great care must be taken that the diagnosis is absolutely accurate, and that an atypical form of central positional nystagmus does not explain the failure to respond to appropriate management.

Not infrequently, an appropriate understanding of vestibular pathology, knowledge of the range of vestibular disorders, and the rationale for management can lead to appropriate medical management of symptoms that have initially appeared intractable.

OTHER FORMS OF CHRONIC VERTIGO

The management strategy set out above applies most commonly to unilateral peripheral vestibular disorders of a multiplicity of aetiologies: traumatic, infective, and vascular. However it is equally applicable to bilateral vestibular failure of acquired type.

Children with congenital bilateral vestibular failure, caused by either a genetic defect or anomalous inner ear development, compensate extremely well and can ski, swim, and skateboard, provided vestibular loss is an isolated abnormality. If, however, there are other motor or cognitive defects or visual impairment, the effects of congenital bilateral vestibular loss are profound.

Acquired bilateral vestibular failure is most commonly caused by gentamicin treatment, but also can be associated

with trauma, bilateral Menière's disease, or as an idiopathic, presumably late onset, genetic origin. This is extremely disabling in the early stages with oscillopsia and gross ataxia, though intensive vestibular rehabilitation can be highly effective, and should be pursued aggressively.

The most difficult form of chronic vertigo to treat is that caused by central vestibular disorders, such as vascular events, multiple sclerosis, and spinocerebellar degenerations, which may or may not give rise to a variety of central eye movement disorders, including vertical, periodic alternating, seesaw, and pendular nystagmus. Such conditions are commonly associated with pronounced disequilibrium, instability, and profound nausea and vomiting. Regrettably, there is no one specific treatment that is of benefit in all patients. Physiotherapy may have a role to play in educating the patient in gait and posture strategies to maximise ability to walk and maintain a degree of stability, while some centrally induced eye movements can be controlled by medication acting at neurotransmitter sites in the central nervous system. The treatment is empirical, but drugs which have been shown to be effective include clonazepam, gabapentin, baclofen, flunarizine, and barbiturates. Each drug should be titrated against known side effects—for example, muscle weakness for baclofen and sedation for clonazepam. In addition, ondansetron, a potent, highly selective 5-HT₃ receptor antagonist, has proved effective in such patients in combatting nausea, particularly in patients with vertigo caused by brainstem stroke.

CONCLUSION

Dizziness is a symptom which tends to cause "heartsink" in many clinicians and is often dismissed as a trivial symptom. However, there is a wealth of literature highlighting the very significant social and occupational morbidity experienced by patients with balance disorders. For the neurologist, who is frequently presented with a dizzy patient, an understanding of the pathophysiology of balance disorders and appropriate management interventions is key to implementing

appropriate rehabilitation strategies which lead to symptomatic recovery and significant improvement in this large group of patients with low morbidity and few long term sequelae. Perhaps most importantly, an understanding of vestibular rehabilitation prevents patients from being referred from department to department in search of an explanation for their chronic symptoms.

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